

or more than two attacks a day occur, 6 to 8 mg per day of methysergide is recommended. Close clinical follow-up with an awareness of the potential for retroperitoneal fibrosis is necessary. Continuous use of this medication for more than six months without a drug-free interval of three to four weeks is not recommended. If these measures fail, a short course of steroids (40 mg of prednisone) for five to seven days with rapid tapering of the dosage over three weeks induces a remission in 75 percent of patients.

In chronic cluster headaches the best form of therapy is 300 mg of lithium carbonate given two to four times a day, with care taken to maintain a blood level below 1.2 mEq per liter. More than 85 percent of patients respond favorably to this regimen. In the subgroup with CPH, indomethacin (25 to 50 mg three times a day) is very effective.

Acute attacks of either type of cluster headaches are best treated symptomatically with sublingual ergotamine preparations (2 mg) or inhalation of 100 percent oxygen at 5 to 7 liters per minute for ten minutes.

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Lacunar Infarction

LACUNES ARE DEFINED pathologically as small trabeculated cavities (0.5 to 20 mm) found in the deeper parts of the brain such as the putamen, pons, thalamus, caudate and internal capsule. These lesions are a result of ischemic infarction in the distribution of penetrating arteries varying in diameter from 80 to 1,000 μ . Although this lesion and some of its clinical correlates were described before the turn of the century, the syndrome of lacunar infarction was largely ignored by modern clinicians until the exquisitely detailed studies of C. Miller Fisher firmly established the clinical and pathological characteristics of this process. The magnitude of the problem is indicated by the prospective data of the Harvard Cooperative Stroke Registry; 20 percent of all patients entered were diagnosed as having lacunar infarction.

Fisher studied the brains from 1,042 consecutive autopsies and found lacunes in 11 percent. Most of the lacunes were small (1 to 4 mm) and

located in the putamen (37 percent). Ten percent were found in the internal capsule. The lesion was almost exclusively confined to patients with a history of hypertension and strongly correlated with the presence of moderate to severe atherosclerosis. Two types of arterial pathologic conditions are found to underlie lacunes: lipohyalinosis and focal atherosclerosis. Both are essentially unique to cerebral vasculature. Lipohyalinosis affects cerebral arteries 80 to 300 μ in diameter and produces mainly small lacunes averaging 2 mm in diameter which are often found as asymptomatic lesions in hypertensive patients at autopsy. Focal atherosclerosis affects larger penetrating arteries 400 to 1,000 μ in diameter and produces larger lacunes (rarely up to 2 cm in diameter) which are probably responsible for most symptomatic lacunar infarcts. Pathologically, focal atherosclerosis consists of isolated miniature plaques of large fat filled macrophages.

Fisher has described four clinical syndromes resulting from lacunar infarction at different anatomic sites. The most common lacunar stroke (accounting for approximately 50 percent of all such strokes) is pure motor hemiplegia. This is a pure motor syndrome of hemiparesis involving face, arm and leg with associated long tract signs and often dysarthria. The responsible lesion is located in the contralateral internal capsule, pons or medulla (face is spared with the latter). A second syndrome is pure sensory stroke characterized by the onset of paresthesias and mild sensory loss over one entire side of the body with little or no functional disability. The lesion is located in the contralateral posteroventral nucleus of the thalamus or sensory portion of the internal capsule. The third syndrome is ataxic hemiparesis characterized by the development of difficulty in walking and ipsilateral arm incoordination. A mild hemiparesis is present with ipsilateral ataxia of the limbs. The lesion is located in the contralateral basis pontis. The fourth clinical pattern is referred to as the *dysarthria clumsy hand syndrome* and is characterized by moderate dysarthria, facial weakness and clumsiness of one hand. The lesion is in the contralateral pons or internal capsule. Other lacunar syndromes will undoubtedly be described following further clinical-pathological correlation.

Transient ischemic attacks may precede lacunar infarction in a fourth of patients and usually occur within 24 hours of the stroke. Stroke onset is more often gradual or saltatory than sudden. Most patients in whom a lacunar stroke develops can look

forward to moderate to excellent recovery even if their initial symptoms were quite disabling. Because of the unique pathologic features of lacunar infarcts, headache, obtundation, cortical deficits and seizures are notably absent and no electrocardiographic abnormalities are seen. A computed tomographic scan may either be normal or show a small enhancing lesion in the distribution of a penetrating artery, and it is an important diagnostic study since the clinical features alone can be misleading. Using computed tomography, Weisberg studied cases of pure motor hemiplegia and found a variety of causes. Thus, the diagnosis of lacunar stroke is made when the neurologic features fit one of the described lacunar syndromes, headaches and the like are absent, and diagnostic studies are compatible.

Treatment consists of controlling existing hyper-

tension in about 80 percent of these patients; however, blood pressure should be lowered cautiously in acute stroke victims because abrupt decreases may precipitate further neurologic symptoms. Because of the location and size of involved vessels, angiography and vascular surgical procedures are not indicated. Anticoagulation may be beneficial if transient attacks of pure motor hemiplegia are occurring, but prolonged anticoagulation is usually not recommended. The role of antiplatelet agents in such patients has not been assessed.

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